Therapeutic Management of Cerebral Arteriovenous Malformations. Present Role of Interventional Neuroradiology

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Key words: AVMs, embolization, therapeutic neuroradiology

This chapter summarizes the authors' experience in the endovascular therapy of cerebral arteriovenous malformations (AVMs). This clinical series includes 660 patients treated from 1980 to 2005.

The first 148 patients were treated at University Hospital, in London, Ontario Canada, in association with Drs. Allan Fox, Dave Pelz, John Girvin and Charles Drake. The next 512 patients were treated at UCLA Medical Center, Los Angeles, California in association with Drs. Gary Duckwiler, Reza Jahan, Jacques Dion, Pierre Gobin, Neil Martin and John Frazee.

Only patients treated with superselective endovascular/intraoperative catheterization and embolization of avm arterial feeders were included. Cerebral arteriovenous malformations treated by non-selective injection of beads in ICA or vertebral arteries were excluded.

Modern neuroimaging modalities associated to the anatomical, topographic and functional evaluations of cerebral avms such as brain CT and CTA, MRI, MRA and functional MRI are all utilized at UCLA Medical Center. They have become essential in the therapeutic management of avms closely related to cerebral eloquent areas (figure 1).

Patients Age and Sex

The patients'age ranged between 4 and 83 years old, with an age average of 45.7 years old.

Three hundred and fifty four patients were male (53.6%) and 306 were female (46.4%).

Patients Clinical Presentation (table I)

Three hundred and eleven patients presented with an intracerebral hemorrhage (47.1%). Ninety two bleeds were predominantly subarachnoid (13.9%), 170 were parenchymal (25.8%) and 49 patients had a predominant intraventricular hemorrhage (7.4%).

Two hundred and sixty one patients (39.5%) presented with seizures , 42 patients (6.5%) had severe headaches, 33 patients (5%)developed a progressive neurological syndrome and in 13 patients (1.9%) the avm was discovered incidentally.

Size of AVMs (table II)

One hundred and seventy-four AVMs were *small* (<3 cm in largest diameter) (26.3%), 248 were *medium* (3 to 6 cm in largest diameter) (37.5%), 214 were *large* (longest diameter >6 cm) (32.6%) and 24 were *giant* (3.6%), (26.3%).

AVM Angioarchitecture by Superselective Angiography (table III)

Diagnostic imaging modalities such as standard cerebral angiography, head CTA and MRA offer excellent overall information on avm size and location.

Three-D rotational digital angiography is not as useful in cerebral avms as in cerenbral aneurysms. The exception is the avm supplied by anterior or posterior perforators. In these

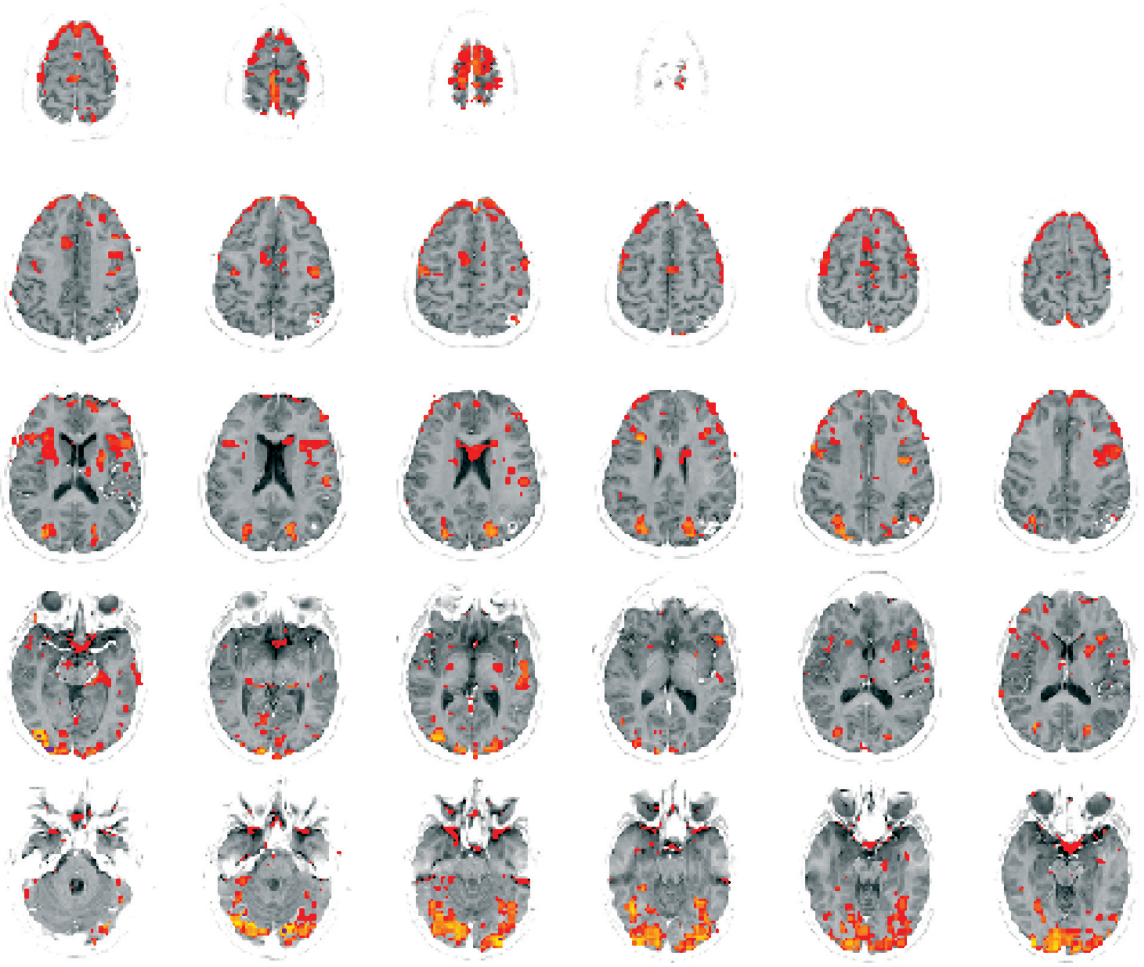


Figure 1 Functional MRI in a patient harboring a small left parietal avm. Specific paradigms were used to depict primary motor-sensory strips and their relationship to the avm nidus.

Table I **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

Clinical Presentation			
Hemorrhage	SAH	92 pts.	13.9%
	Parenchemal	170 pts.	25.8%
	Intraventricular	49 pts.	7.4%
Seizures		261 pts.	39.5%
Progressive Neurological Syndrome		33 pts.	5%
Headaches		42 pts.	6.5%
Incidental		13 pts.	1.9%

Table II **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

AVM Size		
• Small	174 pts.	26.3%
• Medium	248 pts.	37.5%
• Large	214 pts.	32.6%
• Giant	24 pts.	3.6%

Table III **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

AVM Topography			
• Cortical/Subcortical	Dominant	279 pts.	42.3%
	N-Dominant	233 pts.	35.3%
• Cerebellum		48 pts.	7.4%
• Corpus Callosum		37 pts.	5.6%
• Basal Ganglia		27 pts.	4
• Incisura		17 pts.	2.6%
• Thalamus		19 pts.	2.8%

Table IV **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

Techniques of Embolization		
• Transfemoral	599 pts.	90.7%
• Intraoperative	24 pts.	3.6%
• Transfemoral + Intraoperative	8 pts.	1.3%
• Attempts	12 pts.	1.8%
• Functional Evaluation	17 pts.	2.6%

cases, the 3D rotational angiogram identifies the ostium of perforators better than the standard angiogram, facilitating their superselective catherization (figure 2).

Superselective angiography of AVM arterial feeders continues to be the best diagnostic tool to depict anatomical data that influences the type and delivery of embolic agents.

This anatomical data was collected from more than 1200 superselective angiograms performed immediately before delivery of embolic agents into the AVM nidus.

Intranidal aneurysms were found in 109 patients (16.5%) (figure 3).

Intranidal arteriovenous fistulae were identified in 145 AVMs (21.9%) (figure 4).

Stenosis of large AVM draining veins were observed in 102 cases (15.4%) and contribution from anterior or posterior arterial perforators was depicted in 93 AVMs (14%).

AVM Topography (table IV)

Five hundred and twelve AVMs (77.5%) had predominant cortical/subcortical locations. Two hundred and seventy nine (42.3%) involved the dominant and 233 AVM (35.3%) were located in non-dominant cerebral hemispheres.

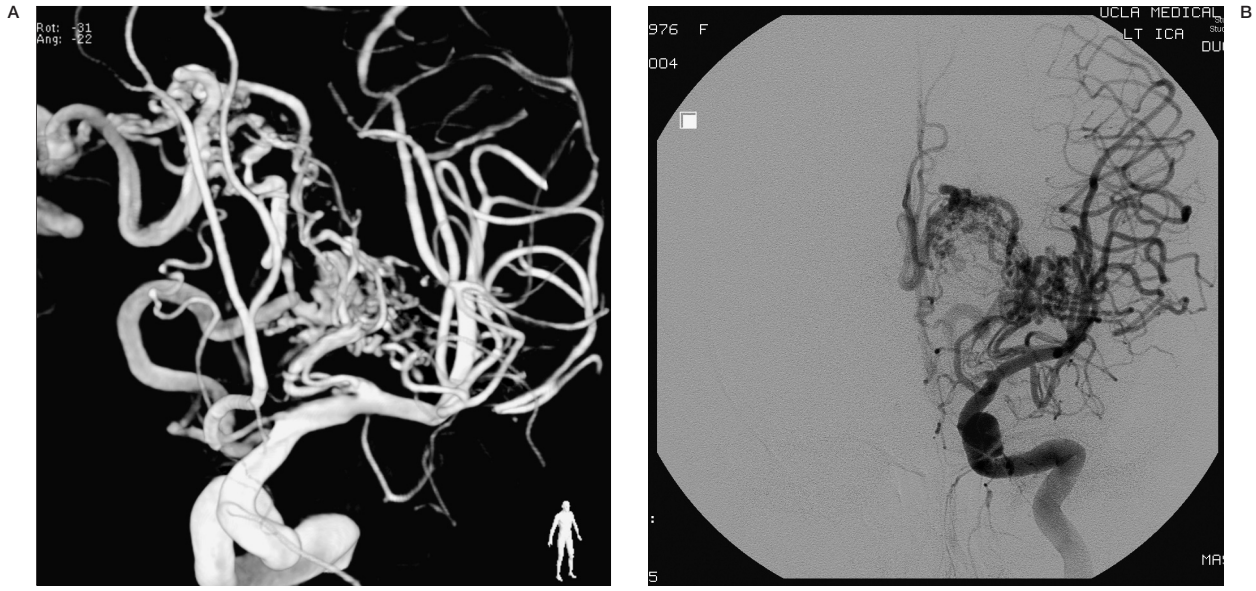


Figure 2 3-D *digital angiography and AVMs*. A)- 3-D rotational digital angiogram shows distinct origin of lenticulostriate arteries. B)- 2D digital angiogram shows the same vascular anatomy though with less details. This difference in visualization of perforators in more complicated cases impinges upon the technical success or failure of their catheterization.

Forty eight (7.4%) AVMs involved the cerebellum, 37 (5.6%) were in the corpus callosum, 27(4%) were located in basal ganglia, 17 (2.6%) were in incisura and 19 (2.8%) involved the thalamus.

Techniques of AVM Embolization

The aim of the endovascular therapist is to occlude as much of the avm nidus with preservation of blood supply of the normal brain parenchyma.

The authors success rate of endovascular complete AVM occlusion is relatively low (56/660 patients-8.5%) though 264/660 (–43.3%) patients had a complete anatomical cure of the avm using a combination of embolization and surgery.

These results are the reflection of a team approach that emphasizes to achieve a complete AVM cure with acceptable morbidy/mortality, in the shortest possible time. The exceptional quality of neurovascular surgery at UCLA allows this type of approach with excellent anatomical and clinical outcomes.

Most patients were under general anesthesia throughout the endovascular embolization.

In patients with AVMs located in brain eloquent areas or fed by arterial perforators (medial lenticulostriate, thalamo-perforators, anterior choroidal artery,etc) a functional amytal testing was performed with the patient awake , EEG monitoring and close neurological monitoring.

The amytal testing consisted in a selective, rapid injection of 36 mg of amytal using a 3 cc syringe while the patient was performing a functional task (elevating controlateral arm or moving toes, counting or repeating a complex sentence, etc).

This test could be repeated if a questionable result was observed).

The injection of amytal was delivered into the arterial feeder approximately 4 cm proximal to the AVM nidus. The target of this functional test was to evaluate the brain parenchyma surrounding the AVM nidus and supplied by the arterial feeder before reaching the AVM nidus (arterial retrograde thrombosis after AVM embolization).

The functional amytal testing was performed in 235 AVMs. (35.6%).

A *positive* amytal testing was observed in 25 cases (10.6%).

A *negative* test was seen in 215 patients (91.4%). Thirteen of those tests that were interpreted as negative were false negative (6.04%).

The selective catheterization of arterial feed-

ers was performed using flow-guided or over-the guidewire microcatheters. The over-the-guidewire microcatheter is particularly useful in feeders related to high flow fistulae because they allow delivery of detachable coils follwed by acrylics. They are also useful to catheterize feeders of slow flow AVMs such as small ones or AVMs compressed by parenchymal hematomas.

All embolizations were preceded by superselective digital angiography of the arterial feeder using a 6 frames per second. This information was used to select the acrylic polymerization time, to depict arteries en passage, intranidal aneurysms and high flow arterial and arteriolo-venular fistulae.

The AVM embolization was performed as close as possible to the AVM nidus and all efforts were made to avoid untoward early occlusion of the AVM draining vein/s. In this series, the most common etiology of postembolization AVM rupture of the last 512 patients was not arterial perforation but untoward abrupt occlusion of draining veins in an incompletely embolized AVM nidus. In our experience at UCLA Medical Center, this is the only reason to perform postembolization emergency surgical resection of a residual AVM nidus.

Systemic heparinization was utilized in all patients by bolus intravenous injection of 3000 units of heparin followed by an i/v bolus of 1000 units per hour. Systemic heparinization was reversed after the procedure with i/v injection of 10 mg of Protamine Sulfate per 1000 units of Heparin. All patients were admitted to Neuro-ICU for 12 hours and then, if indicated, transferred to the Neurosurgery hospital floor.

All patients had a *postembolization non-contrast Head CT scan* to depict untoward embolization of AVM draining veins, dural sinuses and silent intracranial bleeds (small subarachnoid , parenchymal or interventricular hemorrhages due to perforation of a small artery or untoward occlusion of a draining vein in an incompletely embolized AVM nidus). The surgical removal of the residual AVM nidus was commonly performed between 7 to 10 days after embolization. An emergency surgical procedure was indicated only in those AVMs showing venous stagnation related to untoward occlusion of AVM venous drainage.

The transfemoral, transarterial route was used in 599 patients (90.7%). The intraoperative arterial catheterization was used in 24 patients (3.6%). The combination of these two techniques were used in 8 patients (1.8%). In 17 patients (2.6%) a superselective arteriogram followed by a positive amytal testing was performed. These AVMs were not embolized. In 12 patients (1.8%) it was not possible to achieve a safe position for embolization due to very tortuous vascular anatomy.

The intraoperative and combined endovascular techniques were only used in the first 148 patients. The development of new microcatheters and guidewires after 1987 allowed a safer and more distal endovascular transarterial navigation and discarded completely the intraoperative approach.

AVM Embolic Agents (table V)

Isobutyl-cyanoacrylate was utilized in 247 avms (37.4%)

N-butyl-cyanoacrylate was used in 317 avms (48%).

A mixture of Avitene, 30% ethanol and PVA was delivered in 35 patients (5.3%). This embolic agent was abandoned due to the high recanalization rate depicted in angio follow-ups.

Polyvinyl alcohol (PVA) particles were delivered in 17 AVMs (2.6%). This embolic agent was used in association with acrylics in all cases.

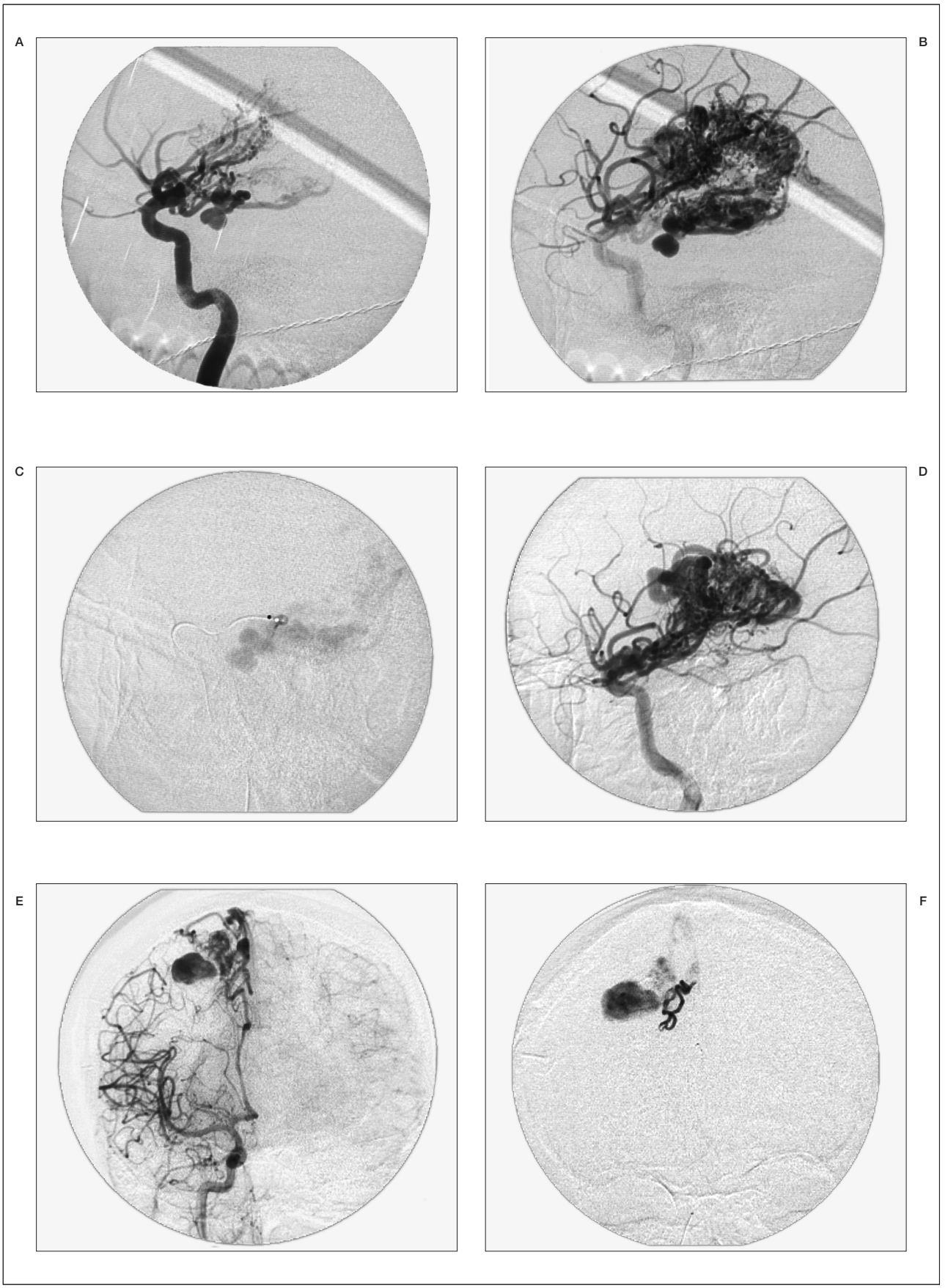
Detachable coils were used in 10 AVMs (1.5%). They were utilized to occlude high flow arteriovenous fistulae associated with an AVM nidus. Detachable coils were used alone or in combination with acrylics to avoid untoward glue migration into AVM draining veins, dural sinuses or lungs (figure 5).

Thirty four AVMs (5.2%) were embolized with Onyx. The use of this new liquid embolic agent has been temporarily discontinued until FDA approval of this product in USA is obtained.

Overall Morphological Results (table VI)

Table VI describes the overall anatomical results. In some cases more than 1 embolization procedure was performed. Large AVMs situated in watershed areas are always supplied by feeders arising from middle, anterior and posterior cerebral arteries. In these cases 2 and sometimes 3 endovascular sessions were performed, with embolization of 3 to 5 pedicles per session.

In 113 cases (17.1%) 50% or less of the AVM nidus was occluded using a single or combination of embolic agents.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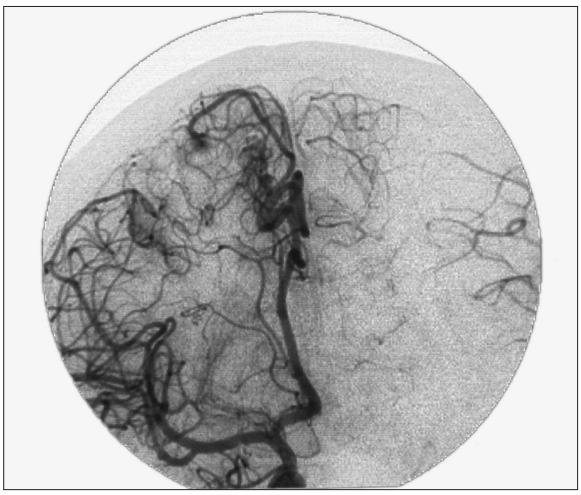

Figure 3 Association of intranidal aneurysms and AVMs. A and B) lateral view of left internal carotid angiogram shows an AVM nidus associated with an intranidal aneurysm arising from the left anterior choroidal artery. This patient presented with an intraventricular bleed most probably related to aneurysm rupture. C) preembolization superselective angiogram of left anterior choroidal artery shows the aneurysm and distal participation of blood supply to AVM nidus. D) postembolization left ICA angiogram shows occlusion of the aneurysm and a small portion of the AVM nidus.

Table V **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

Embolic Agents		
• IBCA	247 pts.	37.4%
• NBCA	317 pts.	48%
• Avit + 30% ethanol	35 pts.	5.3%
• PVA	17 pts.	2.6%
• Detachable coils	10 pts.	1.5%
• Onyx	34 pts.	5.2%

Table VI **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

Morphological Results		
• < 50%	113 pts.	17.1%
• 50-75%	242 pts.	36.6%
• 75-90%	227 pts.	34.4%
• 100%	56 pts.	8.5%
• Surgery + 100%	264 pts.	43.3%
• Radiosurgery	89 pts.	13.5%

Table VII **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

Technical Complications			
Patients	Catheter Glued	Catheter Perforation	Arterial Occlusion
First 148	2 (1.3%)	7 (4.7%)	17 (11.4%)
Next 512	0 (0%)	6 (1.1%)	11 (2.1%)

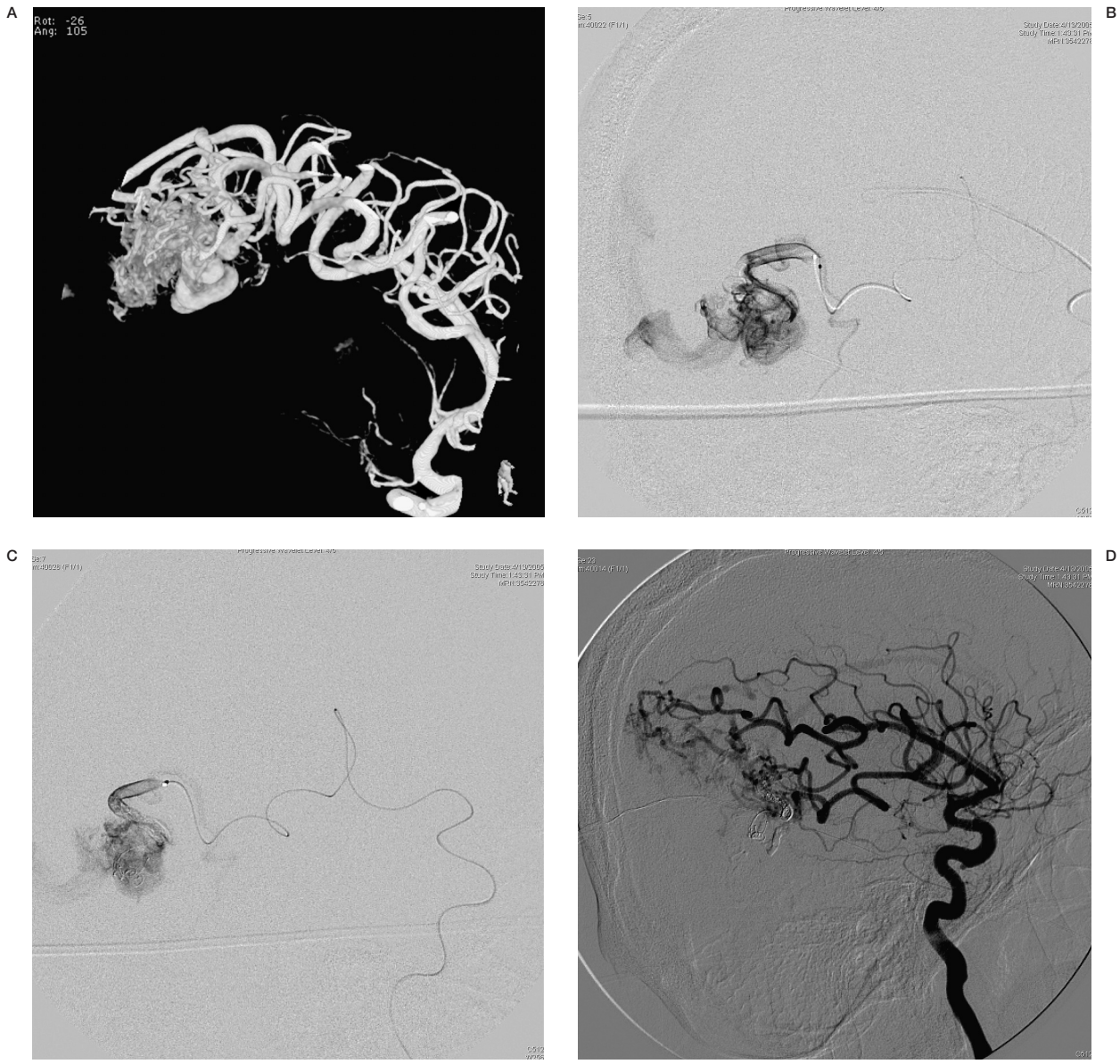

Figure 4 Association *of large arteriovenous fistulae and brain AVMs*. A) 3D rotational angiogram shows a right temporal AVM nidus and a varix probably related to an arteriovenous fistula. B) pre-embolization superselective angiogram of posterior temporal feeders shows a direct arteriovenous fistula draining into the varix. C) selective angiogram shows partial occlusion of the arteriovenous fistula using liquid coils. This arterial feeder was then occluded with an injection of 0.2 cc of 50% acrylic-ethiodol mixture. D) postembolization right ICA angiogram shows complete occlusion of 2 large a/.v fistulae (liquid coils+ acrylics) and filling of AVM nidus. A pre-surgical embolization of the residual AVM nidus will be performed in immediate future.

Table VIII **Therapeutic Management of Brain Arteriovenous Malformations: Experience with 660 Patients**

Immediate Morbidity				
Patients	Mild	Moderate	Severe	Death
First 148 Pts.	14 (9.5%)	6 (6%)	11 (7.5%)	8 (4.5%)
Next 512 Pts.	20 (3.9%)	17 (3.3%)	15 (2.9%)	8 (1.5%)

In 242 cases(36.6%), a 50-75% nidus occlusion was achieved.

Seventy five to 90% nidus occlusion was achieved in 227 pts (34.4%) and 100% nidus occlusion was achieved in 56 pts (8.5%) (figure 6).

In 264 patients (43.3%) embolization was followed by complete surgical removal of the residual AVM nidus. (figure 7). The aim of the presurgical embolization of AVMs included: occlusion of intranidal aneurysms and large arteriovenous fistulae, occlusion of deep arterial feeders such as posterior cerebral, choroidal and lenticulostriate feeders, functional evaluation of potential essential cortical areas with amytal testing, occlusion of cortical AVM as close as possible to AVM nidus to reduce postembolization edema and cortical infarct. The surgical removal of the residual AVM was performed 7 to 10 days postembolization. The only indication for emergency surgery following embolization was the realization of untoward venous occlusion due to acrylics migration into AVM veins and the presence of significant venous stagnation in the immediate postembolization cerebral angiogram.

In 89 patients (13.5%) embolization was combined with stereotactic radiosurgery. In these patients, the AVM nidus embolization was performed with the patient having the stereotactic frame in place. This patient preparation allowed an immediate postembolization radiation therapy of the residual AVM nidus (figure 8) In many cases the stereotactic radiosurgery was planned using the pre-embolization angiographic images. This was particularly true in postembolization angiograms showing patchy occlusion of the AVM nidus without achieving a consolidated and solid nidus size reduction (figure 9).

Technical Complications (table VII)

Only technical complications associated with endovascular procedures were analyzed.

These data were divided in 2 groups: one group included the first 148 patients and a second group included the remaining 512 patients.

In the first group, transfemoral, intraoperative and combined arterial catheterization and embolization were utilized.This group also included the use of the first generation of flow guided microcatheters with inflatable balloons. Only IBCA was used as liquid embolic agent.

In the second group, flow guided microcatheters without an inflatable balloon and over the guidewire microcatheters were used for superselective angiography and embolization.

In these cases a variety of embolic agents were used, depending upon the AVM angioarchitecture depicted from superselective angiography.

These technical differences elicited a very positive impact on the technical complications. Catheters glued in the brain dropped from 1.3% to 0%, arterial perforation dropped from 4.7% to 1.1% and untoward occlusion of arteries supplying normal brain parenchyma decreased from 11.4% to 2.1%.

Immediate Postembolization Morbidity (table VIII)

The noticeable differences observed in the technical complications of those 2 groups are also reflected in the patients immediate clinical outcomes.

Postembolization clinical complications were classified as *mild* (patient could return to his/her personal and professional functional life), *moderate* (able to be independent at home but incapable to return to previous working capacity) and *severe* (patient lost personal functional independence and incapable of working).

In the first 148 patients, 14 patients (9.5%) showed mild neurological deficit, 6 patients (6%) showed a moderate and 11 patients (7.5%) showed a severe neurological deficit. Eight patients died (4.5%). In the following 521 patients, 20 patients (3.9%) showed mild morbidity, 17 patients (3.3%) developed moderate morbidity and 15 patients (2.9%). showed a severe neurological complication.

Eight patient (1.5%) died during or immediately postprocedures.

Nine of the 16 deaths were related to technical complications occurring during AVM embolizations (1.36%) and in 7 patients (1.06%) patient demise was unrelated to procedures (CHF, pulmonary thromboembolism, lung abcess, etc).

Longterm Clinical Outcomes

Information collected on patients long-term clinical outcomes included postembolization and postsurgical patients visits to the UCLA Neurovascular Clinic, neurosurgical follow-up and telephone conversations. Pertinent infor-

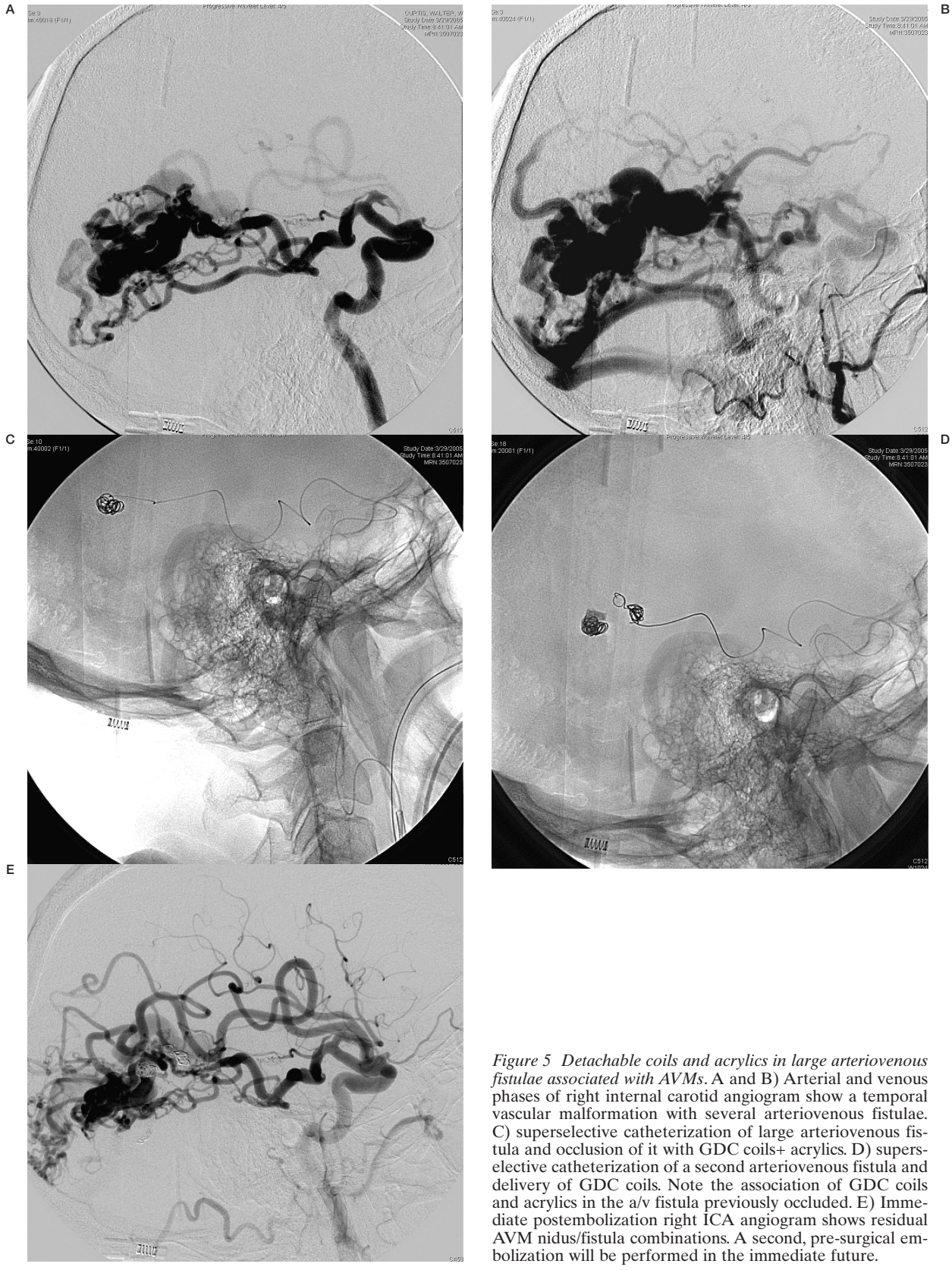

Figure 5 Detachable coils and acrylics in large arteriovenous fistulae associated with AVMs. A and B) Arterial and venous phases of right internal carotid angiogram show a temporal vascular malformation with several arteriovenous fistulae. C) superselective catheterization of large arteriovenous fistula and occlusion of it with GDC coils+ acrylics. D) superselective catheterization of a second arteriovenous fistula and delivery of GDC coils. Note the association of GDC coils and acrylics in the a/v fistula previously occluded. E) Immediate postembolization right ICA angiogram shows residual AVM nidus/fistula combinations. A second, pre-surgical embolization will be performed in the immediate future.

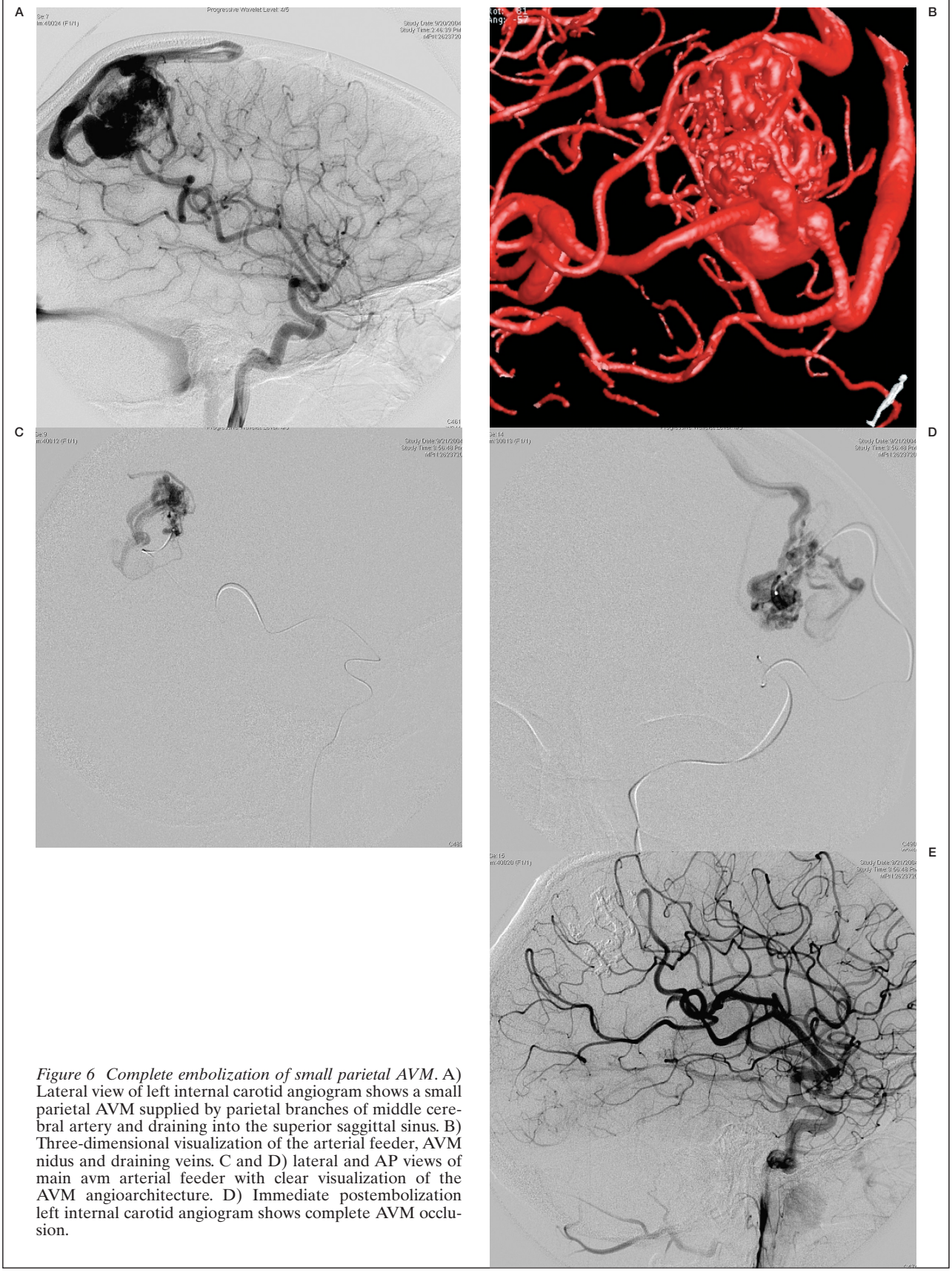

Figure 6 Complete embolization of small parietal AVM. A) Lateral view of left internal carotid angiogram shows a small parietal AVM supplied by parietal branches of middle cerebral artery and draining into the superior saggittal sinus. B) Three-dimensional visualization of the arterial feeder, AVM nidus and draining veins. C and D) lateral and AP views of main avm arterial feeder with clear visualization of the AVM angioarchitecture. D) Immediate postembolization left internal carotid angiogram shows complete AVM occlusion.

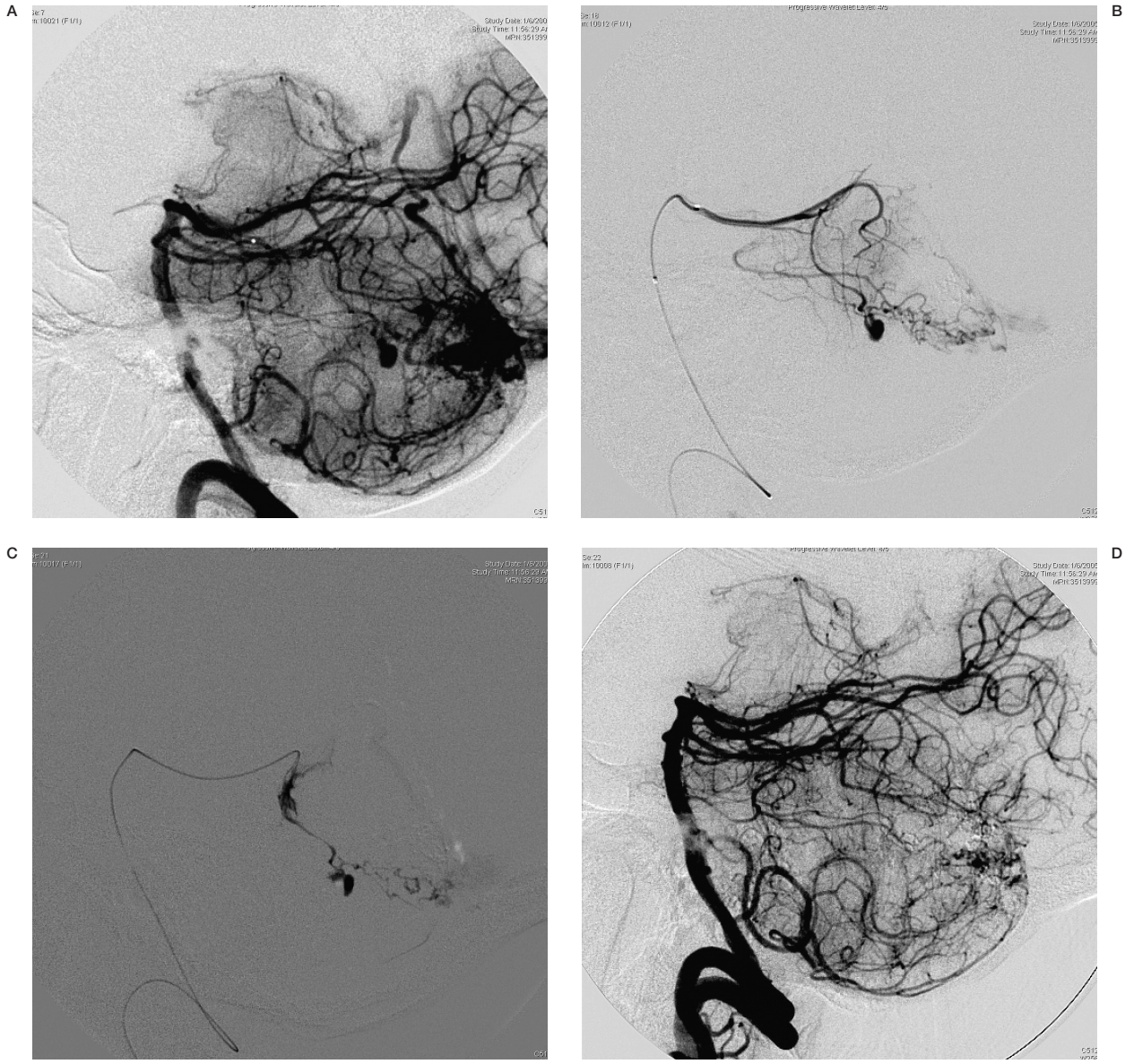

Figure 7 Pre-surgical embolization of cerebellar AVM. A) lateral view of left vertebral angiogram shows a cerebellar AVM associated with a peripheral aneurysm. Note the mass affect related to an underlying cerebellar hematoma. B) selective angiogram of the left superior cerebellar artery shows the peripheral aneurysm and AVM nidus. C) Pre-embolization selective catheterization of artery having the peripheral aneurysm. It was occluded with acrylics injection. D) Immediate postembolization vertebral angiogram shows a small residual AVM nidus and occlusion of the peripheral aneurysm. Successful surgical resection of the residual AVM was performed 3 days later.

mation failed to be collected in 161 patients (24.3%) (patients from abroad, patients changes of local address or migration to other states, patients reluctance to communicate, etc).

Seventy two of the remaining 499 patients (14.4%) had a neurological deficit. The neurological deficit was mild in 37 patients (7.4%), moderate in 16 patients (3.2%) and severe in 22 patients (4.4%).

Three patients had a fatal re-hemorrhage 3/311-0.96%) from large, incompletely embolized avms (8 months, 14 months and 21 months postembolization).

We could not identify late hemorrhages in

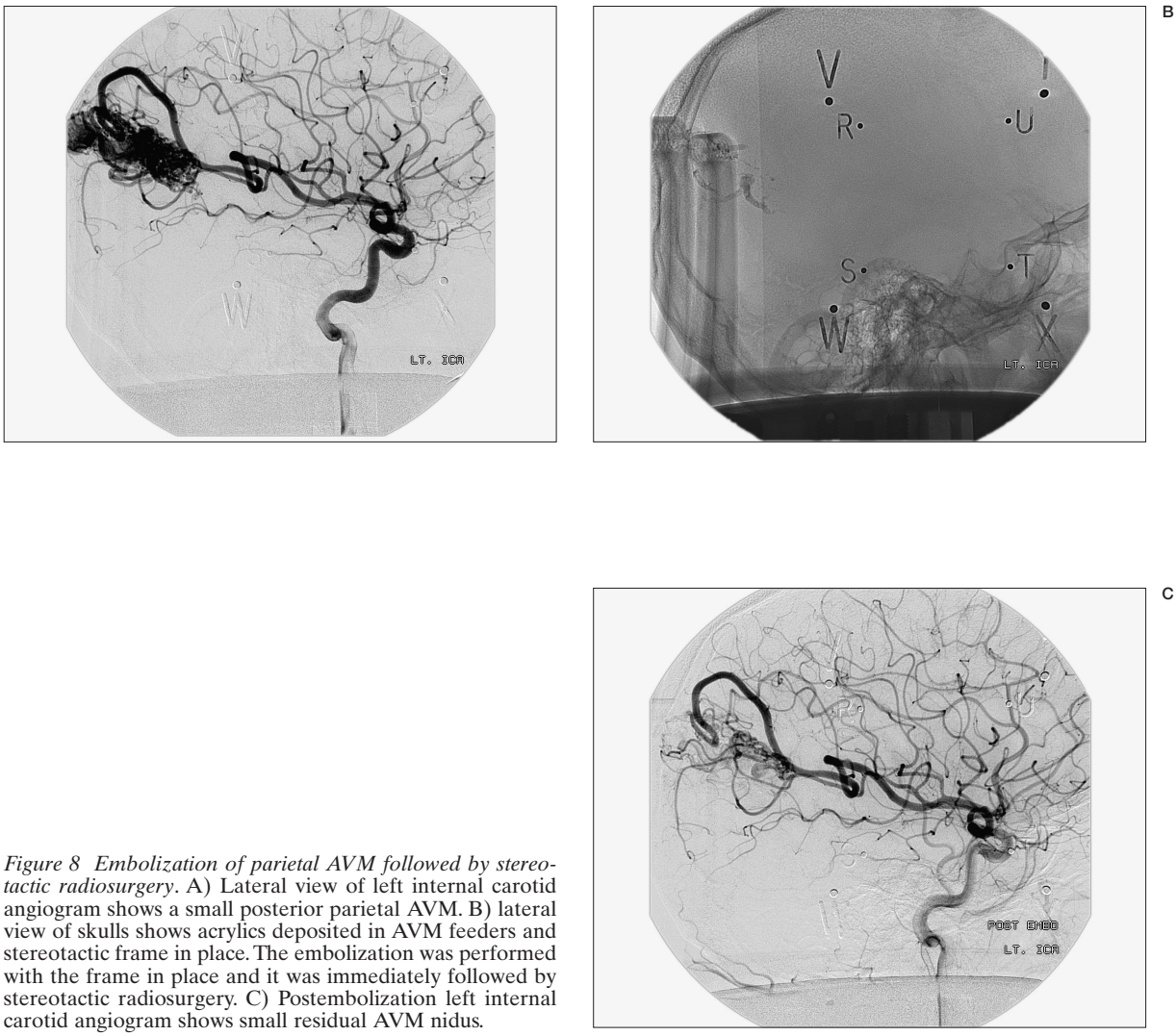

Figure 8 Embolization of parietal AVM followed by stereotactic radiosurgery. A) Lateral view of left internal carotid angiogram shows a small posterior parietal AVM. B) lateral view of skulls shows acrylics deposited in AVM feeders and stereotactic frame in place. The embolization was performed with the frame in place and it was immediately followed by stereotactic radiosurgery. C) Postembolization left internal carotid angiogram shows small residual AVM nidus.

patients presenting with seizures or progressive neurological deficit.

We did not achieved successful embolization in most large or giant AVMs presenting with progressive neurological deterioration.

In 24 of 33 patients (74%) we failed to reverse neurological deficit and change the progressive neurological deterioration. In four large cerebellar AVMs we may have succeeded in stopping the progression of cerebellar truncal ataxia but no reversal of pre-embolization cereberllar sigs was observed.

We did not observe increased technical and clinical morbidities or death in these cases. To-

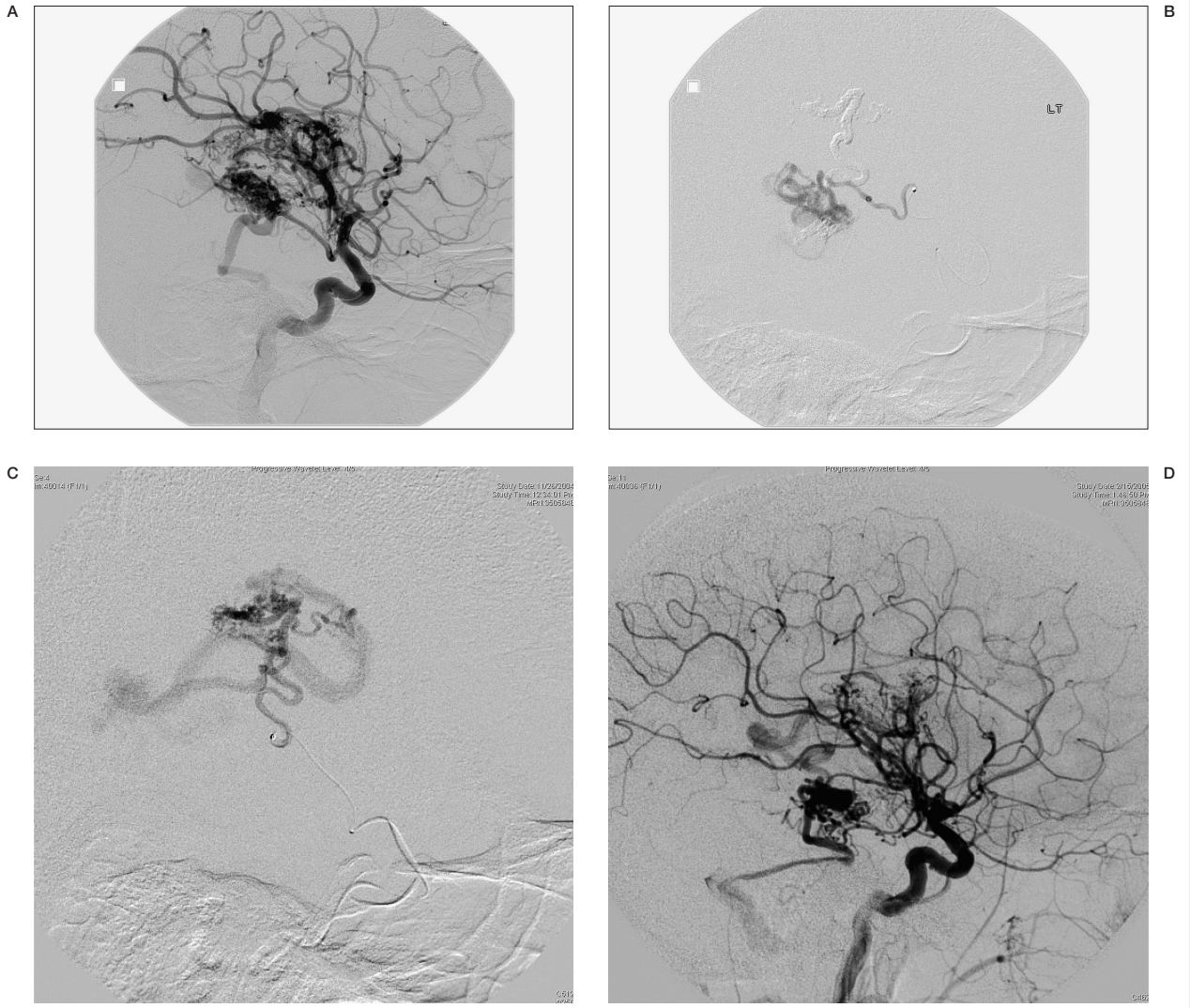

Figure 9 Patchy occlusion of AVM nidus. A) Lateral view of left internal carotid angiogram shows a patchy deep AVM supplied by anterior choroidal artery and lenticulostriate perforators. B) Selective catheterization and embolization of anterior choroidal feeder. C) Selective catheterization and embolization of lenticulostriate feeder. D) Immediate postembolization left internal carotid angiogram shows reduction in size of the AVM nidus and 2 patches of residual AVM.

day we embolize these type of AVMs if we identify large arteriovenous fistulae associated with AVM nidus. In these cases we can observe dramatic local and regional hemodynamic changes that can be achieved with very high success rate and low technical/clinical morbidity.

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